

Evaluating the Unconscious Patient

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What is consciousness?

In broadest terms, it is an awareness of self and the environment. Coma is the most dramatic of the disorders of consciousness, but it is only the end point in a continuum. Any disease process that can cause coma may initially present with mild alterations of, and progressively decreasing, mental status. Careful serial observations and accurate communication of these findings is imperative in the evaluation and treatment of these patients.

Consciousness has two components: content and arousal. Content is controlled by the cerebral hemispheres (also called cerebral cortex, or grey matter). It is marked by purposeful motor function and the use of language.

Content defines **awareness** of the environment. Because of the extensive reserves of the grey matter, diffuse bilateral cortical dysfunction is required to produce a coma. Any form of speech, even unintelligible grunts, signifies some cortical preservation. This area of the central nervous system is most sensitive to metabolic disturbances, such as drug overdoses, or metabolic acidosis.

Arousal is level of consciousness. Patients can be **awake** without being aware - the vegetative state that we see, for example, in patients that have survived brain hypoxia with diffuse cortical injury. However, they must be awake to be aware - that is, content cannot exist without arousal.

What is coma?

It is the absence of any psychologically understandable response to external stimuli or inner need. However the nomenclature traditionally used to describe decreased level of consciousness suffers from inexact definition and frequent inappropriate application. Coma, stupor, obtundation, lethargy, all describe a dysfunction of wakefulness that is difficult to define with exactness.

Coma is probably best described as that altered state of consciousness in which the patient lies quietly with eyes closed in the presence of environmental stimuli. It is at the end of the spectrum of decreased levels of consciousness.

Somewhere before that lies “stupor”, “obtundation”, “lethargy”, and “semicoma”. Acute confusional state usually refers to an inability to think with normal speed and clarity. Delirium describes a state of alteration of both the level and content of consciousness characterized by hyper alert and hyper autonomic irritability, agitated motor movements, disorientation, and vivid hallucinations, as seen in alcohol withdrawal, or stimulant drug intoxication.

The better alternative to using these imprecise terms to specify a patient’s abnormal mental state is to describe behavioral responses to specific stimuli. The “AVPU” system commonly taught in paramedic school is very useful in this respect. (A - Awake and Aware; V - reaction to Verbal stimulus; P - reaction to Painful stimulus; U - Unresponsive to stimuli).

One very useful system of observations for grading level of consciousness that employs the AVPU grid is the Glasgow Coma Scale (GCS). The ability to repeat and accurately describe mental status is of tremendous benefit in patient care, as responsibility is passed from EMS to ER.

The simplicity of the GCS belies its neuroanatomic complexity. The three components of the GCS query the functional integrity of different parts of the central nervous system. Eye opening is a brainstem function. Speech is a cerebral cortical function. Motor response involves both the brainstem and the cortex.

Causes of Coma

Intracranial structural lesions

Trauma
Spontaneous bleed
Thrombosis/embolus
Infection with abscess
Mass

Extracranial insults

Toxins
Metabolic abnormalities (eg. DKA)
Environmental insults (eg. Heat/cold)
Infection without mass effect

What are the causes of coma?

- ▶ Intracranial structural lesions such as those obtained by trauma, spontaneous bleed, thrombosis, embolus, infection or mass.
- ▶ Extracranial causes include toxins, metabolic abnormalities, and environmental insults.

The differentiation of cause is important in the care of the patient with coma. Where are the clues? The state of consciousness (AVPU, GCS), especially the skeletal motor response, with attention to any focal or lateralizing findings; respiratory patterns; pupils; ocular reflexes; and general appearance and surroundings.

The simple evaluation of AVPU, repeated at short intervals, and accurately recorded, can be easily and consistently communicated. Reactions to verbal, painful and other stimuli gauge the depth of coma and provide a window into sensory and motor function in the comatose patient. It is important to note the response of even a slight mumble, for the capacity to speak even a few words indicates a high level of brain functioning.

GCS is also of value, especially in trauma, where the numerical score has been correlated with prognosis. Focal or lateralizing findings during this assessment of motor function are an important indication of structural lesions, rather than toxic or metabolic problems. Quick evaluation of the ability to speak normally, and the ability to use facial muscles contributes to this differential (Cincinnati stroke exam). Motor system evaluation includes an assessment of the patient for hemiplegia, and depends on detecting asymmetry of movement and muscle tone. Reduced spontaneous motion on one side of the body may only be appreciated by serial observation.

How do you check for hemiplegia in a comatose patient?

In acute lesions of the CNS, muscle tone is usually absent. This is best observed with an arm dropping or leg dropping maneuver.

When both arms are held above the patient and forearm is released, the hemiplegic wrist drops at right angles while the unaffected side, the side with some remaining tone, falls more slowly. A similar maneuver may be repeated in the lower extremities. The legs are elevated slightly, and first one leg is extended at the knee, then the other. They are dropped in turn. A difference in tone may cause a visible difference in the rate of fall. Other clues to hemiplegia include observation of the movement and puffing of cheeks with respirations; flaccid facial muscles on the hemiplegic side puff out. Opening the eyelids and releasing them simultaneously usually results in the slower closure of the eye on the hemiplegic side of the body. The orbicularis muscle on the normal side retains muscle tone and closes more rapidly than that on the paralyzed side.

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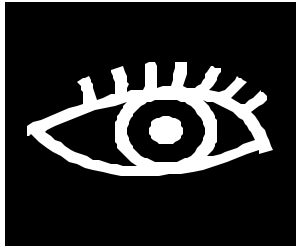
A patient in lighter stages of coma may display a variety of subtle movements. There may be voluntary acts, such as turning in bed, pulling up sheets, and other purposeful appearing movements. Attention should be directed to any asymmetry of movements that might indicate the presence of a hemiparesis. Repetitive movements of any kind should be carefully evaluated for possible seizure activity. A rhythmic twitching of the mouth or an eyelid may be the only motor correlate of seizure. Lip smacking or chewing movements may appear purposeful but indicate seizures. Spontaneous swallowing, yawning or sneezing indicates a light state of coma. Other movements such as shivering, quick gross motions (myoclonic jerks) or twitching of muscle fiber groups, are most often seen in diffuse insults to the brain such as anoxic or metabolic encephalopathy.

Deeply comatose patients may be recognized by their unnatural position; a comfortable appearing patient with legs crossed or curled up usually has an intact brainstem and thalamus. Although posturing has been associated with anatomic location, a strict correspondence between these postures and anatomic locuses of structural damage does not occur. These postures can be produced by a variety of lesions in a variety of locations. However, it is considered a focal finding, and change indicates progression of a lesion and possible pending herniation.

Pupillary reactivity is classically preserved in coma of toxic-metabolic origin, even when all other neurologic function has ceased. It may be difficult to appreciate in drug-induced comas such as those caused by ethanol or barbiturates, or opiates, which typically cause small pupils. Large pupils suggest anticholinergic intoxication or presence of drugs such as cocaine and amphetamines. Glaucoma eye drops can also cause this.

A strong light stimulus to one eye should elicit brisk and consensual pupillary constriction. Sometimes this response is difficult to illicit in bright ambient light - but is it one of the most useful distinguishing characteristics in coma. No pupil should be called unreactive until ambient light has been reduced and pupils have been rechecked. Even more important is asymmetry of pupillary responses. In the absence of previous

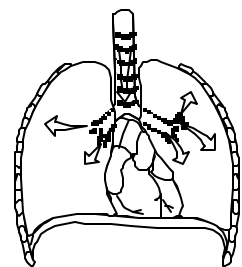
ophthalmologic surgery, an artificial eye or other local ocular disease, pupillary response should be equal. If not, a structural lesions should be strongly suspected.



Slight disconjugate gaze may occur in a variety of causes of coma, and often resolves with stimulation. A conjugate lateral gaze preference can be seen in seizures and focal lesions. Sustained downward deviation may occur with brainstem damage, subarachnoid hemorrhage, or hypoxic and metabolic encephalopathy. Sustained upward gaze (sundowning) is usually the result of severe encephalopathy. Sustained upward gaze is usually the result of severe encephalopathy. Brainstem or peripheral damage usually results in nonconjugate gaze.

Respiratory patterns can vary in rate and rhythm. Necessary airway control should never be delayed in order to assess the patient's pattern of breathing. Respiratory arrest may occur transiently in many conditions other than brain death. Overdoses such as sedatives, narcotics, tranquilizers, alcohol and barbiturates are common examples. It may also accompany subarachnoid hemorrhage or other structural intracranial catastrophes, and rarely associated with endocrine disorders. Sudden apnea may occur with posterior fossa mass lesions. Since it occurs transiently in many causes of coma, absence of respiratory effort is of no prognostic value when the cause of coma is uncertain.

Rapid breathing, tachypnea, has many causes and indicate a problem but is less useful for differentiating between the many causes of tachypnea. These include increased metabolic rates from fever, thyroid toxicity, exertion, or infection. Other reasons include stress; pain; cardiac dysfunction; abdominal distention from obesity, ascites or pregnancy; acute respiratory insufficiency from COPD (with respiratory acidosis), pulmonary infiltration, infection, pleural disease, pneumothorax, pulmonary edema, pulmonary embolism, pulmonary contusion. Metabolic acidosis from uremia, DKA, lactic acidosis, paraldehyde ingestion or poisonings with amphetamines, salicylate, methyl alcohol, ethylene glycol, or cocaine are also causes of rapid breathing. Also, encephalopathies from liver or renal failure, anemia or shock from any reason, CNS dysfunctions (early in many causes), such as CVAs can cause rapid respiratory rate, as can psychogenic causes.



Other respiratory patterns are frequently discussed. Cheyne-Stokes respirations occur as the ventilatory effort varies in depth and rate in crescendo, decrescendo pattern that is interspersed with periods of apnea. It occurs with bilateral hemispheric lesions, metabolic abnormalities, hypoxia, CHF, and high altitude. Several other rare and bizarre respiratory patterns were thought to have some localizing value (apneustic - prolonged inspiratory hold; ataxic - irregular rhythm and depth). However, these have also been described with hypoglycemia, anoxia, or meningitis.

In summary, respiratory patterns may be varied. Lesions specific to different parts of the central nervous system have been associated with particular respiratory patterns, however in practice these observations are difficult to make, inconsistent, and for this reason are of limited value.

History provides the most useful information to identify

why the patient is unresponsive, yet that history is generally unavailable because of the patient's condition. After stabilization and during examination, all possible historical clues must be gathered. Field information is critical. Empty pill bottles, information from family and friends, appearance at the scene with smells, potential toxins and potential trauma. Time course of onset can distinguish between many etiologies. Vascular catastrophes are sudden, while metabolic problems such as DKA will develop over hours or days. Prior headache with or without focal symptoms, development of new focal symptoms, nausea and/or vomiting with abrupt loss of consciousness raises the possibility of a potentially salvageable patient who has a subarachnoid or cerebellar hemorrhage. Past medical history is invaluable. Diabetes, cancer, renal or hepatic failure, medic alert badges, all contribute significantly to diagnosis and care. Several patients with similar symptoms may suggest an environmental toxin such as CO poisoning. Extreme heat or cold in the environment may be the primary cause.

Clues to Diagnosis of the Patient with Decreased Level of Consciousness

History

Age

Surroundings

Physical signs and symptoms



Age of the patient may point you to specific causes. For example, in an infant, infection, trauma, and metabolic derangements are most commonly the cause of decreased mental status. In toddlers, infection, trauma, accidental ingestion, and metabolic derangements are common. In a child or adolescent, we begin to see purposeful toxic ingestions added to the list. In adults, we will begin to see CVAs more commonly. In the elderly, the most common causes are CVA, trauma, infection, metabolic disturbances, and toxic ingestions.

Evaluation of the **surroundings** is critical, as has been alluded to above. Empty pill bottles, extremes in temperature, evidence of mechanism of injury in trauma, determination of length of disability based on condition of home, and many more insights are available only to the prehospital provider. By being observant, the prehospital care provider may be able to narrow the list of possibilities tremendously.

Look for evidence of life threatening trauma or systemic disease which are treatable. These include profound shock, hypercarbia/hypoxemia from respiratory failure; presence of moderate to severe hypertension. Severe hypertension is usually CNS. Certain classes of toxins can produce mild to moderate hypertension. Hypotension does not usually result from a CNS injury until the premonitory state. Hypertension plus bradycardia (Cushings reflex) suggests CNS structural disease with increased intracranial pressure.

General Approach - Care for the Patient with Decreased Level of Consciousness

ABCs, Vital Signs, Stabilization
 Trauma?
 Temperature?
 Any evidence for other diseases?

Body temperature is an important vital sign in the comatose patient. Sepsis, hypothermia, and hyperthermia are commonly seen, and treatment can begin immediately. Certain toxins (eg anticholinergics) cause temperature abnormalities as well.

Look for evidence of any other diseases. The appearance and feel of the skin may give clues to the etiology. Is the skin

hot, cold, wet, dry? Are there track marks or old hesitation marks? Rash or petechia? Is there a dialysis shunt? Compartment syndrome or blistering from sustained immobility? Smell the patient's breath for evidence of DKA or other metabolic problem, or toxic ingestion. Are there old surgical wounds? A pacemaker? An indwelling foley catheter?

The difficulty of accurate diagnosis in the suboptimal clinical setting of the prehospital environment is compensated for by following a standardized procedure that begins with safe stabilization of the patient. Patients with nontraumatic conditions such as hypoglycemia or stroke may have fallen and injured themselves secondarily.

The management of the patient with coma is a combination of intensive supportive care and therapeutic interventions specific to each disease process. Intracranial pressure therapy includes hyperventilation, mannitol, and IV steroids. Status epilepticus, CO poisoning, cyanide poisoning, eclampsia, DKA, hyper or hypoglycemia, hypertensive encephalopathy, respiratory failure, hyperthermia/hypothermia, tricyclic overdose, and hyperkalemia, are just a few examples etiologies with specific therapeutic interventions.

Destination criteria is determined by suspected etiology. If trauma is suspected, proceed to a trauma center. If this is a pediatric patient, the choice should be a center with a dedicated pediatric staff. Confusion concerning destination criteria in the comatose elderly patient frequently revolves around the question of which came first, the decreased mental status or the fall to the ground.

In these instances it may be prudent to transport to a trauma center, and it is not necessary to call a "trauma alert" during transport. All local trauma centers will have CT scan immediately available, and can summon the appropriate health care professionals in a timely manner without rolling out the entire trauma team.

In the case of elderly patients found on the ground unconscious with etiology unclear, it is prudent to transport to a trauma center; however, it is not necessary to "trauma alert" these patients.